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THE STATUS OF BOTULISM AS A WORLD HEALTH PROBLEM

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SYNOPSIS

The author reviews the international literature and brings up to date the published statistical data on botulism—a disease which, owing to its spectacular nature and high case-fatality rate, appears to occupy a place out of proportion to its frequency as a cause of death in some regions.

Without exception, botulism is caused by carelessness in the preparation and preservation of vegetable and animal foods. Local customs of eating such food uncooked, in the form of salads, watery conserves, poorly cured or inadequately smoked pork and salted fish products create the botulism problem.

The risk of botulism exists wherever the telluric incidence of Type A, B, and E spores is high. Surveys to appraise the extent of *Clostridium botulinum* in the soils of Asia and Africa are urgently needed so that the magnitude of the potential problem in these areas can be properly evaluated.

Food processors must not relax in the use of properly calculated thermal processes now available for every food commodity. Agencies that promote or give instruction in preservation of food in the home should be thoroughly familiar with the most effective, practical, and inexpensive methods of preventing botulism.

Interest in botulism, or Kerner's disease, which declined between 1930 and 1946, has been renewed recently by numerous publications on human and animal intoxications in France, by the increased frequency of Type E as the causative anaerobe in Alaska, Canada, France, and Japan, and by the desire of epidemiologists and bacteriologists to analyse the scattered factual knowledge of the disease for the benefit of public-health authorities interested in food sanitation.

Prompted by tragic outbreaks of botulism due to consumption of factory-preserved fruit, vegetables, and fish, studies were carried out between 1919 and 1926 in the USA and the extent of this food intoxication

was carefully measured. It was appreciated then that the spectacular nature and high case-fatality rate in the USA gave botulism a place out of proportion to its frequency as a cause of death. Through the searching epidemiological inquiries during that period, some improperly diagnosed cases which had escaped notification were unmasked as botulism, but even so the unusually numerous outbreaks (15 in 1921, 23 in 1922) never exceeded 65 cases with 48 deaths per annum. Largely as a result of the continuous flood of publicity, which called attention to the possible danger inherent in inadequately sterilized or preserved food products, the annual number of reported and confirmed cases of botulism in the USA has since that time remained below 75.

Statistical data on botulism in other countries, with the exception of those compiled in Germany at regular intervals in connexion with the analysis of so-called " meat poisoning ", refer to the relatively few tragic group-intoxications described in clinico-epidemiological reports. Since interest in botulism as a human disease waned during the thirties, records collected in Germany between 1933 and 1938 [57] escaped inclusion in general reviews of this form of poisoning.

Incidence of Human Botulism

The compilation of reports in Table I, tediously culled from the international literature, brings up to date previously published statistical data.[47, 52, 93] It is doubtless incomplete and perhaps repetitious, and may contain errors. The diagnosis in many instances was made on clinical grounds alone, and the disease is not notifiable in many regions. But the figures are telling and re-emphasize that this interesting type of food poisoning in man is, so far as is known, significantly present mainly in certain countries of Europe and North America. This conclusion, however, may be misleading, as recent observations in France and Japan indicate.

Before 1940 in France only 24 authentic cases were attributed to botulism, but during the German occupation the incidence made a surprising upsurge. This was followed by a subsidence. The episodes aroused the interest of a group of investigators at the Institut Pasteur in improving diagnostic procedures and elucidating the ecology of the different types of *Clostridium botulinum* in France.[72] The incidence in France has now settled down to a level similar to that in Germany and Russia.

The average incidence of annual episodes in Germany for a period of 25 years has remained fairly constant, varying from 9 to 15 outbreaks with 30 to 40 victims and carrying an average case-fatality rate of from 17.4% to 30%. For more than a century before the Revolution botulinic syndromes were recognized in Russia.[33, 45] Erratic reporting stressed the fish-borne outbreaks. Later figures compiled under the new régime with

TABLE I. GEOGRAPHICAL DISTRIBUTION, INCIDENCE, AND NATURE OF BOTULINOGENIC FOOD

Country	Period	Source of figures	Number of episodes	Number of cases	Number of deaths	Type of botulinogenic food (confirmed)	
						animal products	vegetable products
Europe							
Austria-Hungary	1891-1909	Compiled	4	14 (?)		4	
Belgium	1894-1906	,,	3	38	3	3	
Czechoslovakia	1921	Sieber [82]	1	2	0		
Denmark	1906-46	Dyggve	4	13	3	4	
,,	1951	Pedersen [69]	1	6	0	1 (fish)	
France	1875-1936	Compiled		24	3		
,,	1940-44	Légroux et al.[40]	500 (approx.)	1 000	15	163 (93 % pork products)	6
,,	1945-48	Verge [93]	85				
,,	1950-54	Compiled	5	26	2	5 (5)	
Germany	1898-1923	Knorr [37]	77	344	60	74	3
,,	1929-31	Meyer [54]	33			33	
,,	1933-38	,, [57]	153	414	41	153 (14)	1
,,	1943-47	,, [58]	18	53	10	18 (38)	
,,	1948	,, [59]	10	33	2	7 (3 fish)	
,,	1949	Farchmin [20]	9	13 (?)	13	6 (fish)	
,,	1923-48	Verge [93]	357	950	119	307	
Great Britain	1922-54	Compiled	7	15	13	7	
Italy	1903-22	Pitini; [71] Pisani [70]	2	5	1	2	(1)
Netherlands	1935-54	Compiled	5	12	2	5	
Norway	1934-42	,,	4	10	3 (?)	3	
Poland	1920-26	,,		20	14		
Sweden	1933-49	,,	4	11			
Switzerland	1885-1932	,,	3	36	5		
Russia	1878-91	,,		288	111		
,,	1818-1913	Matviev [43]		609	283	fish poisoning	
USSR	1917-26			52	35		
,,	1937	Zavadovskaya (quoted by Dolman & Chang [13])		168	56		
Yugoslavia	1916	Novotný & Ringel [65]	1	1			
South America							
Argentina	1920-26	Pando [66]	2	8+	7+	3	
North America							
Canada	1919-54	Dolman [12]	14	63	35	3 (meat) 5 (fish)	6
USA	1899-1954	Meyer & Eddie[52]	514	1 350	861	38 (meat) 28 (fish) 8 (milk)	381
Australia	1948	Gray [26]	2	32	7		2
Japan	1954	Matsui [44]	6	25	10	6 (fish)	

its improved programme of food sanitation show that the number of outbreaks and deaths has fallen off (Zavadovskaya, quoted by Dolman & Chang [13]).

Over the past 50 years isolated cases or small group-intoxications, never exceeding a total of 40 persons with case-fatality rates of 1.5%-15% (except in the Loch Maree tragedy in Scotland, where all 8 victims eating duck paste died [41]), have been reported from Argentina, Austria-Hungary, Belgium, Czechoslovakia, Denmark, Great Britain, Italy, the Netherlands, Norway, Sweden, Switzerland, and Yugoslavia.

Whether these records reflect the actual incidence of botulism in these countries remains conjectural, but these reports contrast sharply with the nearly complete absence of confirmed human cases in Asia and Africa. Botulism was suspected in two cases in India [90] and since 1951 has been proved in Japan (Matsui,[44] and Nakamara, quoted by Matsui [44]). Its existence in the latter country was anticipated when clams, factory-preserved in Japan, caused 4 fatal intoxications (Type B) in the USA in 1936, and a single case there three years later. The apparent immunity of Australia was broken in 1942.[26]

Interest in botulism in the USA—beginning in 1894, growing with an episode at Stanford University in 1913, and flaring up in 1919 through a series of outbreaks attributed to factory-preserved olives and spinach—reached its culmination between 1919 and 1925. A temporary decrease in cases was followed by another rise to the unusual incidence of 26 outbreaks involving 71 persons (43 deaths) during the years of economic depression. Energetic preventive measures, both educational and legal, brought about a decline both in outbreaks and in interest in placing on record newer observations. Only periodic questionnaires to State departments of health [52] and more recently the *Communicable Disease Summaries* of the National Office of Vital Statistics have supplied some picture of the national morbidity and mortality due to botulism. The 514 single and group intoxications are well documented, but recent re-checking has shown that during the periods of diminished interest, even bacteriologically proved cases may have escaped the attention of health agencies. Repeated cross-checking has eliminated many errors. Two revelations in the USA studies warrant particular emphasis: (1) in approximately 10% of the listed intoxications the causative food was not determined, and (2) bacteriological proof—i.e., demonstration of the toxin or the anaerobe responsible for the spoilage of the food—was furnished in only 166, slightly less than a third, of the clinically recognized episodes. Even in California, with an epidemiological intelligence service attuned to botulism, the causal toxin or type of anaerobe was proved in only 69 (33.9%) of 203 outbreaks. For the years 1949 to 1954 inclusive, the actual number of outbreaks recorded were 6, 4, 13, 8, 7, and 5, with a maximum of 25 cases and 10 deaths in 1951.

Vigorous and productive interest in botulism in Canada resulted in the report of 14 episodes, involving 63 persons of whom 35 died; 6 of the cases were bacteriologically proved.[12]

This sketchy analysis projects the fact that throughout the world during the past 50 years approximately 5635 persons contracted botulism, and 1714 of these persons died. The case-fatality rate varies from one place to another and from one year to another. Among the 1350 cases reported in the USA from 1899 to 1954 the rate was 63.7%. In Europe, by contrast, the case-fatality rate has, with few exceptions (Darmstadt outbreak in 1904, 52.3%; Mayer's series reported in 1913—812 cases of which 365, or 44.9%, were fatal), been consistently lower. The rates varied in France between 1.5% and 8%, and in Germany between 10% and 19%.[58]

Obviously the case-fatality rate in a given country will be affected by the extent to which milder attacks are recognized, but the divergences in the prevailing rates are too wide to be imputed entirely to different degrees of diagnostic acumen. Other more plausible explanations have been offered—namely, that the type of *Cl. botulinum* or the nature of the foodstuffs implicated greatly influences the local case-fatality rate. The tendency for Type A strains, so prominent in the American outbreaks, to be more toxigenic (as judged by lethality for mice and primates [8, 28]) appears to favour the first hypothesis. But many Type B toxins administered to primates in an oral dose of 100 mouse LD_{50} (median lethal doses) produce symptoms within 20 hours and death within 24 hours. Furthermore, experiences in France [40] indicate that the gravity of an attack apparently depended not so much on the type of toxin as on the nature of the food, deaths being associated mainly with watery conserved stews, jellied meats, and vegetables. Over 25 years ago it was emphasized that botulinus toxin production was greatly influenced by the nature of the food implicated.[47] The amount of toxin ingested is a critical factor, and this is in turn affected by the physical and chemical characteristics of the food and the method of preserving it. All recent observations support as more plausible the explanation that dissimilarities in the foodstuffs customarily responsible for botulism in various parts of the world (processed meats—especially ham, sausages, and other pork products—in Europe; home-preserved vegetables and fruit in the USA; fish in the USSR) may be major determinants of regional case-fatality rates.

The place of botulism as the cause of death in food-poisoning outbreaks in the Soviet Zone of Germany after the Second World War may be judged by the following figures for 1948:[59] infections and intoxications attributed to meat or meat products involved 2045 persons of whom 12 died, and of these cases only 33 with 2 deaths were attributed to botulism; food not of animal origin caused 1428 cases of poisoning with 42 deaths, but none of these were due to botulism. Because of the status of reporting of food poisoning it is rarely possible to make such a comparison.

Animal Botulism

A brief consideration of animal botulism is deemed essential in order to appraise its importance as a health hazard, either directly through contamination of meat or meat products or indirectly through losses in livestock or food resources. Moreover, the recorded incidence of animal botulism throughout the world furnishes valuable information concerning the geographical distribution of the six types of *Cl. botulinum* (Types A, B, Cα, Cβ, D, and E).

For nearly 50 years it has been known that spoiled, discarded, botulinogenic food can cause the death of barn-yard fowl in rural and urban poultry flocks in the USA and Canada (detailed review in Geiger et al.[24]). As a rule, Type A, and less frequently Type B, toxin has been responsible for the mortality, which reached a peak of 643 chickens in one single episode.[32] Barn-yard ducks, turkeys, and geese also may contract and succumb to botulism. Sporadic deaths of fowl in the barn-yard caused by ingestion of the larvae of *Lucilia caesar* which have fed on carrion contaminated with *Cl. botulinum* Type Cα may also be important.[2, 3]

Also destructive is the mass intoxication of tens of thousands of aquatic wild birds (250 000 in 1932 on the north side of the Great Salt Lake [35]) which annually recur on certain areas of lakes and mud flats in the western States of the USA, Canada, Argentina, Mexico, Uruguay, and Victoria (Australia), and along ponds in zoological gardens in Germany [11] and South Africa.[78] Invariably *Cl. botulinum* Type Cα has been associated with these outbreaks. The conditions which allow formation of the toxin in lakes are now in part understood: the pH is made favourable by seepage of water from alkaline soil and the oxygen deficiency is produced by layers of pond weed (*Potamogeton*), algae, and rotting vegetation; these create ideal conditions for growth of *Cl. botulinum* and toxin formation.[77] The blood of diseased birds may contain the toxin and their livers may quite regularly harbour *Cl. botulinum* Type C; [27] Type A has been found in ducks in the Philippines.[25] Sick birds have been eaten by the indigenous population in New South Wales [21] and by coyotes and ravens along the shores of lakes in the western States of the USA without ill effects.

Mink and other fur-bearing-animal farms in the USA and Sweden have been plagued by serious economic losses as a result of the feeding of improperly handled mammalian or fish feed in which *Cl. botulinum* Type A or, more frequently, Type C has developed its toxin.[60, 75] In Sweden 21 outbreaks occurred between 1950 and 1953, and in Minnesota a single episode entailed a loss of $75 000.[76]

An excellent summary dealing with botulism in domesticated animals observed since 1912 in Australia implicates cereal hay and chaff, grain, harvesting refuse, meadow hay, decayed vegetation, silage, peanut straw, paddy melon soiled with decomposing grasshoppers, rotting potatoes, and carrion

of sheep, cattle and rabbits. The incidence is reflected in the report that during the summers of 1932 and 1933, 100 000 sheep are considered to have died of botulism in Western Australia.[1]

One of the most serious economic and veterinary problems of the South African, and to a lesser extent the Tasmanian, cattle farmer is botulism or " lamziekte ". It is estimated that 100 000 cattle die yearly of this disease in the Union of South Africa.[84] Theiler and his co-workers demonstrated the interesting relationship between botulism and phosphorus deficiency in grazing cattle reared on phosphorus-deficient veldt. These animals, as the result of the deficiency, crave bones of carrion. Because Types C and D are widespread and grow readily under suitable conditions, carrion may become toxic, as may the afterbirth within 24 hours of being expelled from the cow. Botulism is at its worst when climatic conditions favour toxin development, rather than when phosphorus deficiency is at its height.

It is not generally known that, of all carrion, decaying tortoises provide the most favourable conditions for the development of *Cl. botulinum* toxin. The dead bodies of these animals are ideally suited for anaerobic growth; the spongy inner shell becomes saturated with toxin, and the small size of this reptile allows rapid drying and preservation of the toxin.

It is indeed peculiar that, although every piece of carrion examined in some districts has been very toxic and although some Africans will freely eat decomposing meat, outbreaks of human botulism have not occurred in South Africa.[84] These epidemiological observations that the per os toxicity of Types C and D toxin for man cannot be high are amply supported by quantitative tests on primates.[28] The toxin-producing *Cl. botulinum* has been readily found in the bones and meat of decomposing carcasses, in fly larvae, and in the soil underneath or near carcasses, but in over 100 soil samples collected at random in various parts of Vryburg District, where lamziekte is common, the specific anaerobe could not be isolated. More important are the observations that the anaerobes were present in the digestive tract of cattle which had died from lamziekte or *other* causes, in the rectal contents of healthy cattle (in 7 out of 92 animals examined), and in decomposed guinea-pig carcasses when all possibility of contamination from the soil outside had been excluded.[80] Equally noteworthy are the isolations of Types A and B from the liver or intestinal tract of cattle suffering from haemoglobinuria in Nevada, USA; [24] and, more recently, the isolations of Type B (in two cases) and Type A (in three cases) from beef livers collected in the slaughterhouses of Paris.[72] The presence of spores in catgut prepared from the intestines of Spanish sheep and in a leg of mutton obtained in France proves that this herbivore may be a carrier. The spleen of a horse which died of " grass sickness " in Scotland,[89] and the bone-marrow and intestinal contents of healthy hogs [16, 47] and of fresh-water fish (Types A, B, and E) [13, 73] have yielded toxic cultures of various types. It is rather surprising that in the light of these observations

systematic studies have not been instituted to determine the extent of the " silent carrier stage " of *Cl. botulinum* spores in hogs in European countries, where doubtless germination of the anaerobe in inadequately cured hams frequently leads to diffusion of the toxin through the foramina and hence to an island-like poisoning of the muscle flesh near the bone.

That potentially botulinogenic spores are carried in organs and tissues of healthy food animals is irrefutable, and in all probability these spores are not infrequently the source of the poison in meat or meat products responsible for human botulism when the anaerobes belong to Types A, B, and possibly E. These findings render most difficult the biological diagnosis of equine, bovine, ovine, caprine, porcine, and canine botulism, so freely diagnosed in recent years in Belgium, France, Algeria, Spain, and other areas.[93] There is ample evidence that feed soiled with toxic carrion (particularly carcasses of rats and cats) or ingestion of preserved or fermented foods containing the toxin of *Cl. botulinum* may cause botulism in horses and mules,[88, 93] hogs, and other animals. The epizootiology in these instances is quite similar to that of lamziekte, except that ingestion of the toxic feed is accidental, not conditioned by aphosphorosis of the animals.

In a recent very important report from New Jersey, USA, *Cl. botulinum* toxin Type B was for the first time demonstrated in soy-bean ensilage (protein content, 20.4% dry weight) treated with sodium bisulfite. The outbreak affected 45 valuable Angus cattle. Sixteen days after the first feeding of the ensilage an illness, consisting of constipation, weakness of the legs, and paralysis of the tongue, developed and was diagnosed clinically as botulism. Nineteen pregnant cows died; calves and non-pregnant cows reacted less severely, but all were constipated and somewhat weak. Five animals *in extremis* were treated with 100 ml of antiserum, but this did not prevent their death. The remaining ensilage was examined and found to contain 5 minimum mouse lethal doses of *Cl. botulinum* toxin Type B per gram (V. R. Kaschula & G. M. Dack—personal communication, 1955).

When toxin can be demonstrated in suspected feed and when adequate serum therapy exerts a curative effect, the diagnosis of equine or bovine botulism is most likely to be correct. However, most reports on animal botulism, often called forage poisoning, are based solely on clinical evidence. It is wise to recall that the Botulism Commission of 1920-22 accepted the clinical diagnosis of seasonal summer outbreaks, involving up to 3000 horses, in the USA as botulism,[24] but etiological studies carried out since 1930 have conclusively proved that the equine disease was due to arthropod-borne viruses of encephalitis and not to botulism. A thorough comparative study conducted by workers familiar with both equine botulism and western equine encephalomyelitis led to the conclusion that symptoms alone are an insufficient basis for the differential diagnosis of botulism and equine encephalomyelitis.[31] In the course of the study, epizootiological observation

proved valuable in this respect; botulism affects simultaneously several animals on the same feed, whereas equine encephalomyelitis usually affects only one or possibly two horses at a time without any direct feed connexion.[10]

Public-health workers confronted with the task of appraising the incidence and economic significance of intoxications in domesticated animals have no difficulty in accepting the reports on limberneck, " duck disease ", lamziekte, and certain outbreaks in horse stables and dairy herds in France and Belgium, but they are often unable to estimate the magnitude of the losses due to botulism and their impact on human welfare. Because the mortality may be high,[72] the episodes on farms may be serious and must be prevented. Animal botulism is still a problem to be solved.

Distribution of " Clostridium botulinum " in Nature

Cl. botulinum is found all over the world, its natural habitat being the surface layers of virgin soil; it is present also in cultivated and other soils. The early investigations on its occurrence and distribution [49] have now been extended to some countries previously not included in the surveys (Table II).

The low incidence of Types A and B in soils of the British Isles has been confirmed.[29, 42] An equally low level of contamination of soil with Type A and a very slightly higher level with Type B have been found in Sweden.[19] Type A was the only toxigenic anaerobe isolated in the USSR, although the intestinal tract of large sturgeon-type fish yielded, together with Type A (5%), Type B (11%).[45] Type A has been found in Japanese, Australian, and Indian soils, and Type E was isolated in pure culture from a 1-2 g sample of soil from a " victory " garden in western Canada [14]—an outstanding achievement. The finding of Type B in South African soil specimens [36] amply attests to the world-wide distribution of toxigenic types readily demonstrated by relatively crude methods. The surveys give the impression that the organisms are by no means evenly distributed. It is frequently overlooked that the percentage of toxic cultures may be as high as 40-50, but typing or successful isolation of pure culture may be achieved in only 5%. Some enrichment cultures doubtless contain mixtures of the longer-known types, but some may have contained the less thermo-resistant Types C, D, and E. The failure to find these types in early studies in the USA is partly explained by the small size of samples taken from areas where animal botulism is common (alkaline lake shores, chicken yards with carrion, and such places).

The absence of Types C and D from the soil, except underneath or very near decomposing carcasses,[78, 80] in contrast to the relative frequency of these types in the viscera of healthy animals and, more frequently, in decomposed meat and carrion, deserves brief consideration. Failure to demonstrate *Cl. botulinum* in a sample of soil by the methods thus far

TABLE II. ISOLATION OF CL. BOTULINUM FROM THE SOIL SINCE 1922

Number of samples	Number positive	Type	Material	Area	Reference
12	8		Soil from First World War battlefield	Germany	Zeissler & Rassfeld [95]
100	2	A	Soil	British Isles	Leighton & Buxton;[42] Haines [29]
100	1	and			
256	1	B			
111	1	A	Soil	Sweden	Fåhraeus [19]
111	4	B	Soil	Sweden	
240	6	A	Vegetables, fruit, garden soil	Dnepropetrovsk, Russia	Sajaz [79]
150	3	A	Soil		
69	2	A	Sea water	Sea of Azor	
49	2	A	Sea slime	Sea of Azor	Burov et al.[4]
717	1	A	Soil	Japan	Takashi [86]
183	4	A	Soil	Victoria	Australia [1]
160	11	A?	Soil	New South Wales	Eales & Turner; [15]
	2	B	Rabbit carrion		Gray [26]
	2	B	Silage		
		C	Vegetable matter, hay	Tasmania, South Australia Victoria, Western Australia	
	3	D	Soil Wildfowl Pig carrion		
8	4		Soil	India	Pasricha & Panja [64]
1	1	E	Garden soil	Western Canada	Dolman & Kerr [14]
57	2	B	Soil	Cape Province	Knock [36]
12	1	B	Soil	South-west Africa	Knock [36]
62	8	A	Soil	Maryland	Damon & Payabal [9]
62	25	B	Soil		
62	5	C	Soil		
283	26	A	Soil and dust	New York State	Parry [67]
283	5	B			
152	1	A & B	Soil	Georgia	Morse et al.[61]
604	10	B	Soil	Illinois	Jones & Tanner [34]

devised is no proof that the organism is absent. It means merely that the toxin was not present in demonstrable quantity. This may be due to an antagonistic effect of other organisms in the samples (e.g., to destruction of the toxin by an organism such as *Cl. sporogenes*[7]), or to the presence of too few spores when, for example, the sample of soil examined is moist. It is also to be remembered that vegetative cells of *Cl. botulinum* or its spores if of low resistance to heat would be destroyed, along with non-spore-forming cells of other species, by the preliminary heating of the inocula to 80°C. The few dependable heat-resistance studies dealing with the non-ovolytic Types C, D, and E indicate that even heavy suspensions of spores rarely survive exposure for 20 or 30 minutes at 100°C; in fact, most spores may be destroyed in even less time at lower temperatures. The toxins of Types C, D, and E have been readily demonstrated in enrichment culture when, owing to the nature of the source of the sample, it was not necessary to heat it for long periods or when the numbers of fairly resistant spores were large. These conditions are best fulfilled in the liver of birds and cattle and in the intestinal tract or flesh of fish.

The selective localization of the organisms in the animal body deserves further investigation for the purpose of deciding the role of mammals and birds as healthy carriers or as essential sources of the non-ovolytic Types C, D, and E.

Nothwithstanding the difficulties of assessing the prevalence of *Cl. botulinum* in soils, it is accepted that the population of anaerobes in soil within a given area remains fairly constant over long periods [6] and that some relationship exists between the results of the soil surveys and the prevalence of botulism. It can hardly be coincidence that human botulism occurs in California more frequently than in any other area of the USA; the telluric incidence of the anaerobe in California is very high. Nor is it surprising that in California, where Type A is the most prevalent type in the soil, the 69 bacteriologically proved human botulism episodes yielded 59 intoxications (85.5%) with Type A, only 6 (8.6%) with Type B, 2 of mixed Types A and B, and 1 each with Type Cα and E. The Atlantic States, predominantly seeded with Type B, record 7 out of 9 outbreaks as caused by this type ; in Tennessee, with 3 outbreaks, 2 were due to Type B; in Connecticut, Virginia, and Maryland only Type B has been isolated from incriminated food. There is little doubt that the factors which militate against growth and toxin production in cultures of soil also reduce the chances of toxin production in food. The incidence of human botulism is more closely related to the observed percentage of soils producing toxic cultures than a cursory examination of the facts would suggest.

At the moment the only exception to this conclusion must be made for *Cl. botulinum* Type E; surveys so far conducted are too limited to indicate the extent to which this organism occurs in soils. The apparent absence of botulism from Japan, because there are presumably no indigenous

Cl. botulinum spores, is now rudely contradicted by recent outbreaks in which human beings served as pointers to the presence of Type E spores in the soil and littoral waters. Similar local conditions probably prevail in Alaska, France, Denmark, Yugoslavia, Canada, and elsewhere, and it must now be recognized that Type E is widespread in certain parts of North America (Canada) and probably the USSR.

Moreover, it must be recognized that the risk of human botulism exists throughout the world, not merely in Europe and North America. With continued improvement in etiological laboratory diagnoses, botulism will be recognized in Asia and South America. Food processors, and particularly agencies that promote better nutrition and give instruction in food preservation (especially home bottling and home canning), must insist or continue to insist on the use of properly calculated thermal processes, adequate pickling, acidification, or other safe procedures which will entirely eliminate viable spores or prevent with certainty their germination.[18, 22, 63, 64, 92]

Foods and Types of "Clostridium botulinum" associated with Human Botulism

History relates that the incriminated foods in most earlier cases of so-called "sausage poisoning", or Kerner's disease, were of animal origin, i.e., blood or liver sausage and other made-up meats. Recent outbreaks in Central Europe have implicated other foods of animal origin. They were primarily home prepared and became more or less spoiled, probably owing to careless and insanitary treatment of the raw material before canning or to incomplete impregnation of the meat with antiseptic substances, such as wood smoke, and subsequently inadequately home pickled or insufficiently cooked.

In Germany the following foodstuffs have been implicated: pork products, such as smoked, pickled, or salted hams, sausages, and pork brawn; preserved liver and beef products; potted goose, duck, and other meats (with the exception of horse meat); and salted or pickled fish or other sea food.[57] Fish-borne botulism (from herring steeped in dilute vinegar) has been relatively common in and around Kiel since the Second World War,[94] but Type E has not yet been found in Germany. According to the rather vague descriptions published in connexion with case-histories, only two German outbreaks (1904 and 1943) have been definitely traced to vegetables (string beans containing Type A toxin).[85]

In France during the German occupation, the food responsible for 163 cases was ham—salted and smoked or pickled. In 36 other cases the food contained rabbit (3), duck (1), veal (4), goose (4), pork (10); fish (2), and vegetables (6)—spinach (2), beans (1), asparagus (1), peas (1), and kidney beans (1). The reports [40] stress that over 93% of the 500 outbreaks of

botulism with 1000 cases were in one way or another associated with pork products (see Table I). Later, between 1948 and 1953, canned food (5), ham (2), sausages (2), pâtés (2), and mutton (1) were involved.[72] Vegetable products (6) given some sort of processing in boiling water and then allowed to stand after sealing served as a medium for the specific anaerobe. Three fish-borne episodes were caused by Types B and E.[74]

The food responsible for botulism (Type B) in Belgium was ham.[17] In Denmark, Norway, and Sweden, fish and hams were involved (Type B[43] and Type E[69]). Potted duck-paste (Type A), rabbit-pigeon broth,[62] vegetable and nut brawn (Type A),[39, 87] and steak pie (Type B) have been involved in outbreaks reported from Great Britain. In the few reported cases from the Netherlands, a home-made pie of pigeon and pork (Type B) [5] and ham (Type B) [81] were the toxin-carrying foods. A salmon paste was responsible for 3 cases in Italy,[70] while shallots (*Muscari*) and antipasta of Italian commercial manufacture poisoned with Type B toxin some people in the USA (K. F. Meyer, unpublished data; Koser & Reiter [38]). Fish and fish products (Types A, B, and E) prepared from salted, ungutted sturgeon, carp, bream, or catfish are particularly dangerous, though other animal products are associated with botulism in the USSR.[13] A popular dish, " izushi ", prepared by mixing steamed rice, carrots, and rice-yeast with fish, such as sliced raw sole (3 outbreaks), herring (1 outbreak), dace (1 outbreak), and sea bream (1 outbreak), containing *Cl. botulinum* Type E, is the only food known to have caused botulism in Japan,[44] but clams (Type B) and crab commercially canned in Japan have been responsible for 4 fatal intoxications and 1 mild intoxication, respectively, in the USA. Two episodes in Australia were attributed to commercially canned beetroot.[26]

In the USA under-sterilized or inadequately preserved vegetables and fruit are the most frequent sources of human botulism. The histories of 383 single or group intoxications due to plant products list the following items: string beans (100), corn (53), beets (28), spinach or chard (24), asparagus (24), chili peppers (15), olives (14), peas (12), varieties of beans (11), figs (11), mushrooms (10), tomatoes (10), beet tops (9), apricots (4), okra (4), pears (3), peaches (2), and many other vegetable or fruit products (see details in Meyer & Eddie [52]). Meats or meat products caused 40 outbreaks: sausages (9), blood sausage (2), ham (5), pork products (3), beef products (6), chicken (3), chile con carne (2), and such items as lamb stew (1), beef tamales (1), buffalo meat (1), calf-head vinaigrette (1), pork and beans (1), pickled tongue (1), prepared meat dish (1), head cheese (1), meat balls (1), and seal meat (1). Fish and other sea foods caused 32 episodes through the following preparations: salmon (8), tuna (7), clams (5), sardines (Type A) (4), pickled fish (2), herring (2), crab, trout, salmon eggs, and sprats (1 each). In eight instances milk and milk products—cheese and cottage cheese (Types A and B) (6) and canned milk (2) (1 Type A)—

were proved sources of the toxin. A single fatal intoxication after consumption of commercially prepared spread-cheese known as " Liederkranz type ", prepared in the eastern part of the USA, containing 50 MLD of Type B toxin per gram of product, has recently attracted attention.[53]

Up to 1925, commercially canned and preserved foods manufactured in the USA had been at fault in 43 outbreaks: olives (12), spinach (10), string beans (3), beets (2), corn (1), tomato catsup (1), clams (4), tuna (3), sardines (3), salmon (1), milk (2), and cheese (1). After many years of freedom from botulism due to commercial products, mushroom sauce containing Type E toxin caused botulism in three children, one of whom died.[23] During the past 30 years imported commercially canned foods have been involved in American episodes: antipasta and shallots processed in Italy, clams and crabs from Japan, sprats from Germany (Type E), and smoked salmon from Labrador (Type E).

Of the 14 known outbreaks of proved or suspected botulism in Canada, 6 were caused by vegetables—commercially canned beets (1), home-canned tomatoes (1), asparagus (Type A) (1), beets (1), spinach (Type A) (1), and corn (1); 3 by canned meat products—venison (1), seal meat (1), and beef (1); and 5 by fish products—uncooked salmon eggs (1), canned fish (1), canned salmon (Type E) (1), pickled herring (Type E) (1), and pickled trout (Type E) (1). Western Canada shares with the Pacific and Mountain States of the USA a comparatively heavy telluric incidence of Type A. Type E spores are especially prevalent in the more northerly areas of North America, Labrador and Alaska.

This record continues to support the long-established fact that wherever food is even slightly contaminated with *Cl. botulinum*, from either extraneous or *intra vitam* sources, receives some inadequate preliminary treatment, is allowed to stand for a time, and is then eaten uncooked, it may cause botulism. In the USA most of the recent outbreaks have been due to vegetables home-processed in boiling water and then sealed. Provided the food substance contaminated with *Cl. botulinum* is not too acid (pH below 4.6)[91] or too alkaline (pH above 8.99), and is shut off from free access of air, almost any food seems able to become botulinogenic. Under-sterilization or inadequate curing has, with monotonous regularity, been the prime cause of human botulism. This could be readily avoided if (*a*) all home-canned vegetables and other non-acid food were thoroughly boiled before use, and (*b*) the education of the people in rural areas concerning the risks of botulism were vigorous and more sustained. Spoilage is often so slight that only cooking will volatilize the repulsive spoilage gases and warn the consumer that the contents of the jar or bottle are dangerous. Proper sterilization under pressure has eliminated botulism from commercially manufactured food products. But few, if any, of the people who have been or will be the victims of botulism were or are able to use a pressure cooker, Dehydration[30] or curing in properly prepared brine solutions (10%.

enhanced with 100 to 200 parts per million of sodium nitrate)[83] or acid pickling (at least 2% acetic or citric acid with a pH of 4.0) may, however, be easily carried out by most people. These simple preserving and canning essentials must supplement the standing rule that home-canned food should under no circumstances be eaten or even tasted before it has been cooked.[48] Two hundred and forty-eight intoxications with 189 deaths could have been prevented in the USA if these procedures had been followed.

RÉSUMÉ

Le problème du botulisme a suscité récemment un regain d'intérêt, à la suite de nombreuses intoxications humaines et animales et de la fréquence accrue du type E de *Clostridium botulinum* constatée dans les infections survenues en Alaska, au Canada, en France et au Japon.

Au cours des 50 dernières années, 5635 cas, dont 1714 mortels, ont été signalés dans le monde.

La létalité a varié selon les régions et les années. Aux Etats-Unis, de 1899-1954, le taux moyen de létalité atteignit 63,7%. En Europe, ce taux était nettement inférieur: en France, 1,5%-8%, en Allemagne, 10%-19%. Parmi les explications proposées pour expliquer cette différence, on retiendra l'influence du type de *Clostridium* et la nature des substances alimentaires causes de l'intoxication. Ce dernier facteur paraît jouer un rôle déterminant (conserves de viande en Europe, conserves familiales de légumes et de fruits aux Etats-Unis, conserves de poissons en URSS). L'incidence du botulisme en Europe et en Amérique contraste de façon frappante avec l'absence quasi complète de cas confirmés en Asie et en Afrique, jusqu'à ces dernières années.

Le botulisme des animaux est intéressant à étudier. Il représente des risques pour la santé publique; il entraîne une perte d'aliments destinés à la consommation humaine; sa répartition dans le monde permet de se rendre compte de la distribution géographique des six types de *Clostridium*. On sait que la viande avariée peut provoquer le botulisme chez les oiseaux de basse-cour. Ces derniers peuvent s'infecter en ingérant des larves de mouches nourries sur des carcasses en décomposition. Des infections massives d'oiseaux aquatiques sauvages sont signalées annuellement dans les régions de lacs ou de marais du continent américain ou de l'Australie. *Cl. botulinum* type C α est à l'origine de ces hécatombes. Des *Potamogeton*, des algues, des végétaux en décomposition créent dans ces collections d'eau un milieu anaérobie favorable et les infiltrations d'eau provenant de sols alcalins assurent le pH nécessaire à la formation de la toxine. Le sang des oiseaux infectés peut contenir de la toxine, et leur foie des *Clostridium* type C. Des élevages de visons ont été décimés pour avoir été nourris de viande et de poisson hébergeant des *Clostridium* types A et C. En Australie, des cas de botulisme chez les animaux domestiques ont pu être attribués à la paille, aux déchets de mouture, à la végétation pourrissante, à des carcasses de mouton, de bœuf ou de lapin. Le botulisme (lamziekte), qui cause chaque année la mort de quelque 100 000 têtes de bétail en Afrique du Sud, est considéré dans ce pays comme l'un des principaux problèmes d'hygiène publique vétérinaire. Il est en relation avec la pauvreté des sols en phosphates, qui pousse le bétail à dévorer les os des carcasses rencontrées dans le veldt. Il arrive aussi que les spores soient véhiculées par des animaux sains.

Cl. botulinum se rencontre dans le monde entier, dans les couches superficielles du sol. Il existe une relation entre la fréquence des anaérobies dans les sols d'une région donnée et celle du botulisme dans la même région.

Les viandes, le poisson, les légumes et les champignons soumis à des procédés de conservation insuffisants sont les sources les plus fréquentes des cas de botulisme. Il serait facile d'éviter ces infections en prenant soin, avant de les consommer, de faire bouillir les

légumes ou autres aliments non acides, conservés selon des méthodes familiales. L'odeur nauséabonde des gaz de putréfaction dégagés par la cuisson signale l'avarie.

Il importe que les institutions et organisations qui cherchent à améliorer l'alimentation et enseignent au public les méthodes domestiques de conservation des aliments insistent pour que soient appliquées des températures assez élevées, une acidification convenable ou tout autre procédé qui éliminerait les spores de *Clostridium* ou les empêcherait de germer.

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